



Parental body mass index and blood pressure are associated with higher body mass index and blood pressure in their adult offspring: a cross-sectional study in a resource-limited setting in northern Peru

Rodrigo M. Carrillo-Larco^{1,2}, Antonio Bernabé-Ortiz^{1,3}, Víctor G. Sal y Rosas⁴, Katherine A. Sacksteder^{1,5}, Francisco Diez-Canseco¹, María K. Cárdenas¹, Robert H. Gilman^{1,5,6} and J. Jaime Miranda^{1,7}

1 CRONICAS Center of Excellence in Chronic Diseases, Universidad Peruana Cayetano Heredia, Lima, Perú

2 Department of Epidemiology and Biostatistics, School of Public Health, Imperial College London, London, UK

3 Faculty of Epidemiology and Population Health, London School of Hygiene and Tropical Medicine, London, UK

4 Department of Science, Pontificia Universidad Católica del Perú, Lima, Perú

5 Department of International Health, Bloomberg School of Public Health, Johns Hopkins University, Baltimore, USA

6 Área de Investigación y Desarrollo, Asociación Benéfica PRISMA, Lima, Perú

7 Department of Medicine, School of Medicine, Universidad Peruana Cayetano Heredia, Lima, Perú

Abstract

OBJECTIVES High body mass index (BMI) and blood pressure (BP) are major contributors to the high burden of non-communicable diseases in adulthood. Individual high-risk and population approaches for prevention require newer strategies to target these risk factors and focusing on the family to introduce prevention initiatives appears as a promising scenario. Characterisation of the relationship between BMI and BP among the adult members of a given family merits evaluation. We conducted a secondary analysis of an implementation study in Tumbes, Peru, benefiting from data derived from families with at least one adult offspring.

METHODS The exposures of interest were the BMI, systolic BP (SBP) and diastolic BP (DBP) of the mother and father. The outcomes were the BMI, SBP and DBP of the offspring. Mixed-effects linear regression models were conducted.

RESULTS The mean age of the offspring, mothers and fathers was 29 (SD: 9.5), 54 (SD: 11.8) and 59 (SD: 11.6) years, respectively. Father's BMI was associated with a quarter-point increase in offspring BMI, regardless of the sex of the offspring. Mother's BMI had a similar effect on the BMI of her sons, but had no significant effect on her daughters'. Mother's SBP was associated with almost one-tenth of mmHg increase in the SBP of the adult offspring. There was no evidence of an association for DBP.

CONCLUSIONS In families with adult members, the higher the parents' BMI and SBP, the higher their adult offspring's levels will be.

keywords body mass index, overweight, obesity, blood pressure, hypertension, family health

Introduction

Over the last decades, mortality due to non-communicable diseases (NCDs) has increased globally, with cardiovascular diseases at the top of the list [1]. Worldwide trends show that body mass index (BMI) and obesity rates [2, 3], as well as high blood pressure (BP) alongside with diastolic and systolic blood pressure (DBP/SBP) [4], have increased between 1975 and 2015. In 2015, high SBP and BMI were among the leading contributors to disability-adjusted life-years [5]. The impact of high BMI

and high BP on NCDs is not trivial, and it challenges economies and societies.

The epidemiology of these risk factors – BMI and high BP – is not encouraging in developing countries, where there are more deaths attributable to NCDs than in developed nations [6]. Mean BMI in Peru, a middle-income country, in 2014 was approximately 26.5 kg/m² [3], a population average estimate in the range of overweight. A longitudinal study conducted between 2007–2008 and 2012–2013 among Peruvian rural-to-urban migrants, rural and urban subjects showed that the

obesity incidence in these three groups was 2.3, 0.4 and 2.6 per 100 person-years, respectively [7]. This suggests that obesity rates are to increase particularly in urbanised sites and those undergoing rapid urbanisation. In contrast, starting from a baseline hypertension prevalence of 19.7%, a population-based Peruvian study reported a hypertension incidence of 7.12 new cases per 100 person-years, with different risk estimates according to degree of urbanisation; semi-urban sites exhibited the highest risk [8]. These BMI and BP estimates at the population level reveal a complex scenario for NCDs, yet evaluation of these risk factors at the family level, particularly among its adult members, could signal additional windows of opportunity for prevention initiatives. Interventions at the family level have shown promising results in targeting and improving negative lifestyles related to sedentarism [9, 10], diet and weight [11, 12] of the household's children; however, family health of adult subjects has been understudied. Whilst individual-level lifestyle recommendations to improve cardiovascular health are important [13], new interventions considering a whole-family approach can be proposed to reduce weight. Moreover, family health research contributes to understanding the developmental origins of health and disease hypothesis, which still needs to be acknowledged in clinical practice [14].

Although the intrafamily relationship of health risk factors could be understood on genetic studies, in the era of evidence-based medicine, these are not enough to conclude in favour of (or against) interventions. Observational epidemiological studies, although limited in terms of causality assessment, do provide a first step to describe whether there is a health issue in the study population, worthwhile of intervention. Consequently, we aimed to characterise the relationship between parental BMI, SBP and DBP and that of their adult offspring's.

Methods

Study design and setting

This was a cross-sectional analysis using data of the baseline assessment of a population-based implementation study in Peru; further details about the implementation study can be found elsewhere [15]. The study was conducted in Tumbes, a semi-urban setting at sea level in northern Peru. Figures for 2016 revealed that there were 240 590 people in Tumbes, with a life expectancy of 74.7 years; 18.9% reported not having any health insurance, and 9.8–12.6% of the population was under the poverty line [16].

Study population

The population included in this study is the same one enrolled at baseline in the implementation study; [15] there were no differences between our study population and the one of the trial study. Subjects were enrolled from six randomly selected rural villages. All members of the household aged ≥ 18 years were included in the implementation study. The exclusion criteria comprised having any mental illness that would prevent giving informed consent, self-reported chronic kidney disease and self-reported heart disease [15].

Because of the inclusion criteria in the implementation study, only adult subjects were analysed herein. For this study, we included subjects with complete information on BMI, DBP and SBP. Only one family was included per household (first family in the registry) with at least father or mother, regardless of the number of adult offspring, although each family had to have at least one. Thus, in the analysis, there could have been single-parent families, nuclear families or families with several children. The number of participants included in the study is available in Figures S1 and S2, for DBP/SBP and BMI, respectively; also, these figures show the process conducted to identify family relationships/members; first, we cleaned data looking for fathers, mothers and children within the same house; of these, we only kept those with complete data on the outcomes of interest; lastly, we analysed families with at least father or mother and one offspring.

Variables

The clinical evaluation was conducted by trained field-workers following standardised procedures [15]. BP was assessed three times in a resting position after a five-minute resting period, with at least one minute between measurements. For this study, the mean of the last two readings was used. Weight and height were measured with the participants wearing light clothes and no shoes. All other variables were elicited in a paper-based interview using validated questions, such as those from the WHO STEPS approach [15, 17].

Outcome variables. The outcomes were BMI (kg/m^2), DBP (mmHg) and SBP (mmHg) of the offspring in each family, assessed as continuous variables.

Exposure variables. The exposures were BMI (kg/m^2), DBP (mmHg) and SBP (mmHg) of mothers and fathers at each family. These variables were treated as continuous variables.

Other variables. Other variables included in the study were as follows: village in which the participant lived (categorical variable); sex; age (continuous variable); education level (none/primary, secondary and higher education); physical activity assessed with the short version of the International Physical Activity Questionnaire (IPAQ) and classified as low, moderate or high; [18] heavy drinker was defined as having a hangover or ≥ 6 drinks at one occasion at least once per month (Yes/No); current smoker (Yes/No); if the subject added salt to the food when eating (never or at least some times); and father's wealth index, which is the same for the family, and it is a composite numeric index based on facilities and goods owned by the household [19]. Of these, the following were included in the adjusted regression models: offspring's sex, age, physical activity, educational level, father's wealth index and village in which the participant lives; height (offspring's, father's and mother's) was included when BP was the outcome of interest. Other variables (e.g. parental physical activity) were not adjusted for because of strong correlation between parents.

Statistical analysis

Lineal mixed regression models, with two hierarchical levels (individual and family) and an unstructured covariance matrix, were used to assess whether the BMI, DBP and SBP of the parents were associated with the BMI, DBP and SBP of the offspring. Factors that confounded or modified this relationship were included in a multivariate analysis such as age, sex, education level, height (offspring, father and mother), socioeconomic status (of the family) and study village. Analyses were conducted with STATA v13.0 (StataCorp, College Station, TX, USA)

In a pre-specified analysis, sex of the offspring was included as an effect modifier of the relationship between BMI/DBP/SBP of the offspring and the BMI/DBP/SBP of the father and the mother, separately. To account for the situation where offspring currently have only a mother (94, 21.3%) or only a father (30, 6.8%), two indicator variables of whether the family has current a father or a mother were included in the model. Even though families without a father cannot be used to estimate the effect of BMI/SPB/DBP of the father on the offspring, they can still be used to estimate the effect of the other covariates.

Results are presented as the mean difference, at the population level, associated with a one unit increment in the covariate, conditionally on having a father or a mother. 95% confidence intervals were presented for all estimations. Details about the fitted model are available as Appendix S1.

Ethics

The implementation study was approved by the Institutional Review Boards at Universidad Peruana Cayetano Heredia (Lima, Peru) and Johns Hopkins University (Baltimore, USA). Signed informed consent was obtained from all study participants [15].

Results

Characteristics of the study population

There were 955 subjects who met our inclusion criteria; 438 offspring (253 males and 185 females) were available for analysis along with 237 fathers and 280 mothers. The median size of a family was three members [IQR = 3–4, Range = 2–6]. The mean number of offspring per family was 1 [IQR = 1–2, range = 1–4]. The mean age of the offspring, mothers and fathers was 29 (SD: 9.5), 54 (SD: 11.8) and 59 (SD: 11.6) years, respectively ($P < 0.001$, Table 1).

Family aggregation: BMI

In the adjusted model, father's and mother's BMI was associated with higher offspring's BMI (Table 2). For each additional father's BMI unit, the offspring's BMI increased on average 0.25 [95% CI 0.13; 0.37] kg/m²; there was no interaction between the father's BMI and the offspring sex. The BMI of the mother was associated with an average increase of 0.20 [95% CI 0.10; 0.31] in the BMI of the male offspring, whilst this figure for the female offspring was not significant. This suggests that the mother's BMI has a stronger influence on the BMI of her sons than on the BMI of her daughters.

Family aggregation: Blood Pressure

In the adjusted model, an increase in mother's SBP was associated with a mean increment of 0.11 [95% CI 0.05; 0.18] on SBP of the female offspring. Neither father's DBP/SBP nor mother's DBP/SBP significantly interacted with the offspring sex, suggesting there was no same-sex or cross-sex resemblance between parents and offspring regarding BP.

Discussion

Main findings

In semi-urban resource-limited villages in northern Peru, as the parent's BMI increased, so did the male and

Table 1 Sociodemographic and clinical characteristics of the studied families

	Overall	Father	Mother	Male offspring	Female offspring
Sex					
Female	465 (48.7%)	0 (0.0%)	280 (100.0%)		185 (100.0%)
Male	490 (51.3%)	237 (100.0%)	0 (0.0%)	253 (100.0%)	
Age					
Mean (SD)	44 (17.7)	59 (11.6)	54 (11.8)	29 (9.5)	29 (9.5)
Median (IQR)	44 (27–57)	58 (51–67)	53 (46–61)	26.5 (22–32)	27 (22–34)
Education					
None/primary	370 (38.7%)	150 (63.3%)	183 (65.4%)	23 (9.1%)	14 (7.6%)
Secondary	393 (41.2%)	70 (29.5%)	80 (28.6%)	162 (64.0%)	81 (43.8%)
Higher	192 (20.1)	17 (7.2%)	17 (6.1%)	68 (26.9%)	90 (48.7%)
Wealth index					
Bottom	317 (33.5%)	–	–	–	–
Middle	317 (33.5%)	–	–	–	–
Top	312 (33.0%)	–	–	–	–
Physical activity					
Low	616 (64.5%)	105 (44.3%)	238 (85.0%)	133 (52.6%)	140 (75.7%)
Moderate	254 (26.6%)	93 (39.2%)	38 (13.6%)	84 (33.2%)	39 (21.1%)
High	85 (8.9%)	39 (16.5%)	4 (1.4%)	36 (14.2%)	6 (3.2%)
Heavy drinker					
No	881 (92.3%)	217 (91.6%)	278 (99.3%)	202 (79.8%)	184 (99.5%)
Yes	74 (7.8%)	20 (8.4%)	2 (0.7%)	51 (20.2%)	1 (0.5%)
Current smoker					
No	852 (89.5%)	173 (73.6%)	279 (99.6%)	216 (85.7%)	184 (99.5%)
Yes	100 (10.5%)	62 (26.4%)	1 (0.4)	36 (14.3%)	1 (0.5%)
Add salt					
Never	878 (92.0%)	218 (92.0%)	262 (93.6%)	229 (90.5%)	169 (91.9%)
At least some times	76 (8.0%)	19 (8.0%)	18 (6.4%)	24 (9.5%)	15 (8.1%)
BMI					
Mean (SD)	26.7 (4.8)	26.6 (4.4)	29.1 (4.8)	25.1 (4.1)	25.6 (4.7)
Median (IQR)	26.5 (23.3–29.8)	26.1 (23.4–29.6)	29.3 (25.9–32.4)	24.6 (21.8–27.9)	25.3 (22.0–28.4)
Height					
Mean (SD)	159 (8.8)	164.9 (6.3)	152.2 (5.9)	166.8 (6.3)	155.3 (6.2)
Median (IQR)	159.5 (153.2–166.4)	165.2 (160.7–169.4)	152.2 (148.3–156.0)	167.0 (163.0–171.2)	155.2 (151.8–158.7)
DBP					
Mean (SD)	72.7 (10.3)	75.4 (10.7)	74.0 (10.8)	71.7 (9.6)	68.6 (8.4)
Median (IQR)	71.5 (65.5–78.0)	74.0 (67.5–81.5)	72.0 (66.5–80.5)	71.0 (65.5–76.5)	68.0 (63.5–73.5)
SBP					
Mean (SD)	113.8 (17.0)	120.7 (17.2)	116.0 (19.5)	114.2 (11.9)	101.4 (10.9)
Median (IQR)	111.5 (103.0–122)	119.5 (109.5–129.0)	112.5 (103.0–125.5)	112.5 (107.0–119.5)	99.5 (94.5–106.0)

BMI, body mass index; DBP, diastolic blood pressure; SBP, systolic blood pressure. Wealth index is the same at the household level.

female offspring's BMI; the mother's BMI had a bigger effect on her son's BMI than on her daughter's. As the mother's SBP rose, so did the offspring's SBP. These results suggest a resemblance between adult offspring and parents in BMI and SBP. Although the study population was healthy subjects and the magnitude of the association estimates herein described was small, the overall resemblance should not be overlooked because even within a normal range of BMI and BP, there is a trend towards higher values of these risk factors.

Public health implications

Although restricted by the study design and other limitations, the results may call to implement prevention strategies at the family level to avoid high BMI and BP 'contagion' among members, particularly regarding BMI, for which the association estimates were stronger. The scope of our paper is beyond advocating for a particular intervention, yet there are a few examples that could be used to improve cardiovascular risk factors at the family level. Interventions could target the environment or

Table 2 Association between BMI/SBP/DBP of parents and BMI/SBP/DBP of their offspring using a multilevel mixed-effects linear regression model ($n = 442$; cluster = 308)

	Crude estimates		Adjusted estimates	
	β	95% CI	β	95% CI
Model 1: BMI as outcome				
Father–Female Offspring	0.19	0.07;0.32	0.25	0.13;0.37
Father–Male Offspring	0.20	0.08;0.32	0.26	0.14;0.38
Mother–Female Offspring	0.10	–0.02;0.21	0.11	–0.00;0.22
Mother–Male Offspring	0.20	0.10;0.30	0.20	0.10;0.31
Model 2: SBP as outcome				
Father–Female Offspring	0.06	–0.02;0.14	0.05	–0.03;0.12
Father–Male Offspring	0.05	–0.03;0.12	0.03	–0.04;0.11
Mother–Female Offspring	0.11	0.04;0.18	0.09	0.03;0.17
Mother–Male Offspring	0.13	0.07;0.19	0.11	0.05;0.18
Model 3: DBP as outcome				
Father–Female Offspring	0.10	0.01;0.20	0.10	–0.00;0.19
Father–Male Offspring	0.09	–0.01;0.19	0.09	–0.01;0.18
Mother–Female Offspring	0.06	–0.04;0.16	0.07	–0.03;0.17
Mother–Male Offspring	0.08	–0.02;0.17	0.08	–0.02;0.17

Adjusted by village, age, educational level, physical activity of the offspring and wealth index of the family. For the blood pressure models, height of offspring, mother and father was also included. Each line represents the mean effect in the outcome variable in the offspring that the same variable of the father or mother has. For example, in model 1, Father–female offspring measured the mean change in the BMI of the female offspring given a one unit change in the BMI of the father, adjusting for other covariates. Bold values are statistical significant at a threshold of $P < 0.05$.

subjects in the household, addressing portion size and table/dishware features could foster less food consumption [20], presumably having a weight reduction effect [21, 22]. Although the proposed interventions would target all family members, others can be aimed at some members alone. A recent trial showed that a family-based intervention was non-inferior to a parent-based intervention to reduce weight in the children [12], suggesting that not all family members ought to be actively involved in the intervention.

Comparison with previous results: BMI

Our results showed that higher BMI of both parents was associated with higher BMI of the offspring. Other studies have shown similar results [23–25]. A study in China with 23–24-year-old offspring reported that, as father's and mother's BMI increased, so did the offspring's [23]. An Australian study reached a similar conclusion, but also assessing other obesity indicators (e.g. waist circumference) [25]. Comparable figures were retrieved in the United Kingdom too [24]. Also, some studies have suggested that the BMI resemblance may follow a same-sex pattern: father–son and mother–daughter [24–26]. However, other authors have reported a BMI resemblance between father–daughter [27] and mother–son [28]. Our results suggest that the mother's

BMI greatly influences that one of her sons. If the mother's BMI has a stronger correlation with the son's BMI, it could be because of a biased view that males must be robust as a synonym of good health [29, 30]. These beliefs should be taken into consideration when addressing weight loss in the study population, or others with Latino background [31].

Comparison with previous results: blood pressure

Our results showed that there was a correlation between mother's SBP and that of the offspring; nevertheless, results were not conclusive regarding father's SBP or either parent DBP. Previous reports have yielded more conclusive results [23, 32]. A study in Korea with adolescents revealed a positive BP correlation between parents and children; of note, regarding the dyads mother–son and mother–daughter, the correlation was significant for DBP only [32]. With regard to adults, a study with Chinese Han population revealed that higher DBP or SBP of the father and mother was associated with higher SBP in the offspring [23]. A possible explanation for our discrepant results could be lack of statistical power because some confidence intervals were close to significance. Another possible explanation could come from the profile of salt added at the table, which was not different among family members. Thus, approximately everybody in the

family has the same salt intake. Nevertheless, one limitation of this proposed interpretation is that food consumed outside the house was not analysed. Future studies need to address all possible salt sources in the daily diet.

Interpretation of results

The associations between parents and offspring regarding the assessed risk factors could be due to genetic reasons [33, 34], or to shared environmental features. Johnson *et al.* [24] suggested that the different size effects between mother/father and offspring regarding BMI could be explained by genetic factors, but most importantly by environmental ones, because same-sex transmission of genes is not the commonest Mendelian trait [23, 35]. Then, the same-sex resemblance could be explained by factors such as physical activity or diet profiles. Also, interactions between family members, family functioning and parenting styles [36], as well as social networks between grownup siblings [37] could explain the results because of the effects one family member has on the others. It is worth mentioning that the offspring assessed in this study still live with their parents, and most likely they have all lived together since ever. Because health-related behaviours or lifestyles are learnt throughout their lifetime, prevention strategies should be applied early in life. It has been reported a stronger correlation between adiposity indexes between parents and adolescent offspring, than between parents and children [38]. This could suggest that adolescents have already acquired unhealthy lifestyles, which supports starting prevention strategies at early ages. In addition to this potential resemblance of unhealthy health behaviours, evidence in developmental origins of health and diseases further supports looking for and managing the higher risk of non-communicable diseases early in life [14].

The literature suggests that maternal traits affect equally offspring traits, and with a larger effect than paternal traits [39]. Our results partially challenge this statement because, for example, the effect of mother's BMI seems to be larger in sons than daughters. We hypothesise this is because parenting style as well as cultural background, and the perception that males need to be robust as a synonym of good health, most likely occur in Hispanic backgrounds [29]. Therefore, variability in the effect of father's and mother's traits could depend on (biased) perceptions of weight and health standards. Overall, our results support the findings of previous studies and recently summarised by Devakumar *et al.* [39], suggesting that parental traits influence sons and daughter differently.

Strengths and limitations

This study had some strengths. First, we studied families from the general population in a resource-limited setting in Latin America. Second, because BP is the main outcome of the implementation study from which the study sample was drawn [15], it was assessed thoroughly, and so was BMI. Nonetheless, limitations must be highlighted too. First, the study sample was not enrolled for this study in particular; thus, the results could be underpowered and may not represent the targeted population which could bias the results. However, we still present strong associations suggesting our results are robust and probably alert there is a much greater effect. Likewise, because the original trial did not include people who reported kidney or heart diseases, these individuals were not included in this study. Generalisability of our results to families with these patients may be limited. Second, there could be residual confounding; also, due to data availability, we could not include other potential confounders such as diet profile (e.g. fruits/vegetable consumption). Nevertheless, because we included members of the same family living together, they probably shared a similar diet so adjusting for this variable would not have had a strong effect on the results. In spite of these limitations and the cross-sectional design of the analysis, the results showed a strong pattern that deserves to be studied longitudinally to verify whether parental behaviours or characteristics influence the offspring enough to develop high BMI or BP. Third, we did not have data on non-paternity and we did not conduct sensibility analysis to address this possibility. Devakumar *et al.*, in a sensibility analysis, revealed that with increasing levels of non-paternity, the point estimates of the association between parent and offspring BMI slightly decreased for mothers and scarcely increased for fathers [39]. This signals the potential health relevance of further studying non-communicable diseases at the family level and with different family structures, that is, including non-paternity profiles. Fourth, unlike other studies which focused on complete families (i.e. father, mother and child), our regression models accounted for the absence of either the father or the mother. Rather than a limitation, this procedure improved data efficiency, maximising our sample size without the need to undertake imputation methods. When comparing our results with those of a complete-case analysis (i.e. including complete families), the estimates were very similar.

Conclusions

Higher parent's BP (systolic) and BMI are associated with higher BP (systolic) and BMI of the adult offspring living

in the same family. The family level seems to be a feasible scenario to display prevention strategies to reduce aggregation of high BMI and BP among family members.

Acknowledgements

The authors are indebted to all the participants who kindly agreed to collaborate in the study. Special thanks to all field teams for their commitment and hard work. This study was supported by the National Heart, Lung, and Blood Institute (Project 1 U01 HL114180-01), United States, under The Global Alliance for Chronic Diseases (GACD) programme. CRONICAS Center of Excellence in Chronic Diseases is supported by the National Heart, Lung, and Blood Institute Global Health Initiative under the contract Global Health Activities in Developing Countries to Combat Non-Communicable Chronic Diseases (Project Number 268200900033C-1-0-1). RMC-L has received support as a research trainee from the Inter-American Institute for Global Change Research (IAI CRN3036). AB-O is a Wellcome Trust Research Training Fellow in Public Health and Tropical Medicine (103994/Z/14/Z). VGSyR was funded by the Dirección de Gestión de la Investigación at the PUCP through grant DGI-2015-200.

References

- GBD 2015 Mortality and Causes of Death Collaborators. Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet* 2016; **388**: 1459–1544.
- Stevens GA, Singh GM, Lu Y *et al.* National, regional, and global trends in adult overweight and obesity prevalences. *Popul Health Metr* 2012; **10**: 22.
- NCD Risk Factor Collaboration (NCD-RisC). Trends in adult body-mass index in 200 countries from 1975 to 2014: a pooled analysis of 1698 population-based measurement studies with 19.2 million participants. *Lancet* 2016; **387**: 1377–1396.
- NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in blood pressure from 1975 to 2015: a pooled analysis of 1479 population-based measurement studies with 19.1 million participants. *Lancet* 2017; **389**: 37–55.
- GBD 2015 Risk Factors Collaborators. Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet* 2016; **388**: 1659–1724.
- Yusuf S, Rangarajan S, Teo K *et al.* Cardiovascular risk and events in 17 low-, middle-, and high-income countries. *N Engl J Med* 2014; **371**: 818–827.
- Carrillo-Larco RM, Bernabe-Ortiz A, Pillay TD *et al.* Obesity risk in rural, urban and rural-to-urban migrants: prospective results of the PERU MIGRANT study. *Int J Obes (Lond)* 2016; **40**: 181–185.
- Bernabe-Ortiz A, Carrillo-Larco RM, Gilman RH, Checkley W, Smeeth L, Miranda JJ. Impact of urbanisation and altitude on the incidence of, and risk factors for, hypertension. *Heart* 2017; **103**: 827–833.
- Brown HE, Atkin AJ, Panter J, Wong G, Chinapaw MJ, van Sluijs EM. Family-based interventions to increase physical activity in children: a systematic review, meta-analysis and realist synthesis. *Obes Rev* 2016; **17**: 345–360.
- Holm K, Wyatt H, Murphy J, Hill J, Odgen L. Parental influence on child change in physical activity during a family-based intervention for child weight gain prevention. *J Phys Act Health* 2012; **9**: 661–669.
- Viitasalo A, Eloranta AM, Lintu N *et al.* The effects of a 2-year individualized and family-based lifestyle intervention on physical activity, sedentary behavior and diet in children. *Prev Med* 2016; **87**: 81–88.
- Boutelle KN, Rhee KE, Liang J *et al.* Effect of attendance of the child on body weight, energy intake, and physical activity in childhood obesity treatment: a randomized clinical trial. *JAMA Pediatr* 2017; **17**: 622–628.
- Eckel RH, Jakicic JM, Ard JD *et al.* 2013 AHA/ACC guideline on lifestyle management to reduce cardiovascular risk: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. *J Am Coll Cardiol* 2014; **63**: 2960–2984.
- Heindel JJ, Vandenberg LN. Developmental origins of health and disease: a paradigm for understanding disease cause and prevention. *Curr Opin Pediatr* 2015; **27**: 248–253.
- Bernabe-Ortiz A, Diez-Canseco F, Gilman RH, Cardenas MK, Sacksteder KA, Miranda JJ. Launching a salt substitute to reduce blood pressure at the population level: a cluster randomized stepped wedge trial in Peru. *Trials* 2014; **15**: 93.
- Ministerio de Salud. Centro Nacional de Epidemiología, Prevención y Control de Enfermedades. Tumbes. (Available from: http://dge.gob.pe/portal/Asis/indreg/asis_tumbes.pdf).
- WHO STEPwise approach to Surveillance (STEPS): STEPS Manual. In STEPS Manual. (Available from: <http://www.who.int/chp/steps/manual/en/index.html>).
- Ipaq. Ipaq scoring protocol. (Available from: <Http://www.Ipaq.Ki.Se/scoring.Pdf>).
- Townsend P. *Poverty in the United Kingdom: A Survey Of Household Resources And Standards of Living*. Univ of California Press: Berkeley and Los Angeles, 1979.
- Hollands GJ, Shemilt I, Marteau TM *et al.* Portion, package or tableware size for changing selection and consumption of food, alcohol and tobacco. *Cochrane Database Syst Rev* 2015; **9**: CD011045.
- van Ittersum K, Wansink B. Do children really prefer large portions? Visual illusions bias their estimates and intake. *J Am Diet Assoc* 2007; **107**: 1107–1110.
- McClain AD, van den Bos W, Matheson D, Desai M, McClure SM, Robinson TN. Visual illusions and plate

- design: the effects of plate rim widths and rim coloring on perceived food portion size. *Int J Obes (Lond)* 2014; **38**: 657–662.
23. Hu Y, He L, Wu Y, Ma G, Li L, Hu Y. Familial correlation and aggregation of body mass index and blood pressure in Chinese Han population. *BMC Public Health* 2013; **13**: 686.
24. Johnson PC, Logue J, McConnachie A *et al.* Intergenerational change and familial aggregation of body mass index. *Eur J Epidemiol* 2012; **27**: 53–61.
25. Grant JF, Chittleborough CR, Taylor AW. Parental Midlife body shape and association with multiple adult offspring obesity measures: North West Adelaide health study. *PLoS ONE* 2015; **10**: e0137534.
26. Li Z, Luo B, Du L, Hu H, Xie Y. Familial clustering of overweight and obesity among schoolchildren in northern China. *Int J Clin Exp Med* 2014; **7**: 5778–5783.
27. Naess M, Holmen TL, Langaas M, Bjorngaard JH, Kvaloy K. Intergenerational transmission of overweight and obesity from parents to their adolescent offspring - The HUNT Study. *PLoS ONE* 2016; **11**: e0166585.
28. Fuentes RM, Notkola IL, Shemeikka S, Tuomilehto J, Nissinen A. Familial aggregation of body mass index: a population-based family study in eastern Finland. *Horm Metab Res* 2002; **34**: 406–410.
29. Baker EH, Altman CE. Maternal ratings of child health and child obesity, variations by mother's race/ethnicity and nativity. *Matern Child Health J* 2015; **19**: 1000–1009.
30. Lindberg NM, Stevens VJ, Halperin RO. Weight-loss interventions for Hispanic populations: the role of culture. *J Obes* 2013; **2013**: 542736.
31. Lindsay AC, Sussner KM, Greaney ML, Peterson KE. Latina mothers' beliefs and practices related to weight status, feeding, and the development of child overweight. *Public Health Nurs* 2011; **28**: 107–118.
32. Park HS, Park JY, Cho SI. Familial aggregation of the metabolic syndrome in Korean families with adolescents. *Atherosclerosis* 2006; **186**: 215–221.
33. Stunkard AJ, Harris JR, Pedersen NL, McClearn GE. The body-mass index of twins who have been reared apart. *N Engl J Med* 1990; **322**: 1483–1487.
34. Fox CS, Pencina MJ, Heard-Costa NL *et al.* Trends in the association of parental history of obesity over 60 years. *Obesity (Silver Spring)* 2014; **22**: 919–924.
35. Perez-Pastor EM, Metcalf BS, Hosking J, Jeffery AN, Voss LD, Wilkin TJ. Assortative weight gain in mother-daughter and father-son pairs: an emerging source of childhood obesity. Longitudinal study of trios (EarlyBird 43). *Int J Obes (Lond)* 2009; **33**: 727–735.
36. Kitzman-Ulrich H, Wilson DK, St George SM, Lawman H, Segal M, Fairchild A. The integration of a family systems approach for understanding youth obesity, physical activity, and dietary programs. *Clin Child Fam Psychol Rev* 2010; **13**: 231–253.
37. Powell K, Wilcox J, Clonan A *et al.* The role of social networks in the development of overweight and obesity among adults: a scoping review. *BMC Public Health* 2015; **15**: 996.
38. Halvorsen T, Moran A, Jacobs DR Jr *et al.* Relation of cardiometabolic risk factors between parents and children. *J Pediatr* 2015; **167**: 1049–1056 e2.
39. Devakumar D, Grijalva-Eternod C, Cortina-Borja M, Williams J, Fewtrell M, Wells J. Disentangling the associations between parental BMI and offspring body composition using the four-component model. *Am J Hum Biol* 2016; **28**: 524–533.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Figure S1. Number of subjects included in the study, outcome: blood pressure.

Figure S2. Number of subjects included in the study, outcome: BMI.

Appendix S1. Fitted Regression Model.

Corresponding Author Antonio Bernabé-Ortiz, CRONICAS Center of Excellence in Chronic Diseases, Universidad Peruana Cayetano Heredia, Address: Av. Armendáriz 497, 2do Piso, Miraflores, Lima 18, Perú. Tel.: +511 241 6978; E-mail: antonio.bernabe@upch.pe